

Amendments to the Specification

Please replace paragraph [0001] of the specification with the following paragraph:

[0001] This application is a divisional of U.S. Serial No. 10/179,373, filed June 26, 2002, which is a continuation-in-part of U.S. Serial Nos. 10/035,045 filed January 3, 2002 and 09/897,427, now U.S. Patent No. 6,955,887, filed on July 3, 2001, Application 10/179,373 [[,]] claims priority to Provisional Application Serial No. 60/300,434, filed on June 26, 2001, U.S. Provisional Application Serial No. 60/304,749 filed on July 13, 2001, U.S. Provisional Application Serial No. 60/310,493 filed on August 8, 2001, U.S. Provisional Application Serial No. 60/331,771 filed on November 21, 2001, U.S. Provisional Application Serial No. 60/339,472 filed December 14, 2001, and , U.S. Provisional Serial No. 60/372,090 filed April 15, 2002, and U.S. Provisional Application Serial No. 60/374,143 filed on April 22, 2002, all of which are incorporated by reference in their entirety.

Please amend paragraph [0052] of the specification as follows:

[0052] ~~Figure 16~~ Figures 16A and 16B show that lactisole inhibits the receptor activities of human T1R2/T1R3 and human T1R1/T1R3. Figure 16A shows responses of HEK-Gα₁₅, cells transiently transfected with T1R1/T1R3 (circles) to 10mM L-glutamate and HEK-Gα₁₅, transiently transfected with T1R2/T1R3 (squares) to 150 mM sucrose in the presence of variable concentrations of lactisole. Figure 16B shows fold increases in taste detection thresholds in the presence of 1 and 2 mM lactisole for the sweet taste stimuli sucrose and D-tryptophan, the umami taste stimuli L-glutamate (MSG) and L-glutamate plus 0.2 mM IMP, and sodium chloride. Detection thresholds were determined following the method of Schiffman et al.

Please amend paragraph [0071] of the specification as follows:

[0071] The invention especially includes biochemical assays conducted using cells, e.g., mammalian, yeast, insect or other heterologous cells that express one or more full length T1R receptors or fragments, preferably N-terminal domains of T1R1, T1R2 and/or T1R3. The effect of a compound in such assays can be determined using competitive binding assays, e.g., using radioactive glutamate or IMP, fluorescence (e.g., fluorescence polarization, FRET), or GTPγ³⁵S binding assays. As noted, in a preferred embodiment, such assays will utilize cell lines that

stably co-express T1R1/T1R3 or T1R2/T1R3 and a suitable G protein such as $G_{\alpha 15}$. Other appropriate G proteins include the chimeric and variant G proteins disclosed in U.S. Application Serial No. 09/984,292, now U.S. Patent No.6,818,747, and 60/243,770, incorporated by reference in their entirety herein.